10/069,454

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=> d his

     (FILE 'HOME' ENTERED AT 15:07:47 ON 22 DEC 2004)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 15:08:15 ON 22 DEC
     2004
L1          1454 S RNASE(3A)(T2 OR RH OR M OR TRV OR IRP OR LE OR MC1 OR TP OR O
L2           813 S ACTIN(W)BIND?(3A)ACTIVITY
L3             0 S L1 AND L2
L4        635993 S (INHIBIT? OR REDUC? OR PREVENT? OR TREAT? OR REVERS?)(5A)(CAN
L5             7 S L1(S)L4
L6             0 S L2(S)L4
L7             3 S L2 AND L4
L8        337629 S (PROMOT? OR INCREAS? OR FACILITAT? OR ENHANC?)(6A)(CANCER OR
L9             2 S L1 AND L8
L10            4 S L2 AND L8
L11            3 DUP REM L7 (0 DUPLICATES REMOVED)
L12            4 DUP REM L5 (3 DUPLICATES REMOVED)
L13            2 DUP REM L9 (0 DUPLICATES REMOVED)
L14            1 DUP REM L10 (3 DUPLICATES REMOVED)

=> d bib ab 1-3 l11

L11  ANSWER 1 OF 3  BIOSIS  COPYRIGHT (c) 2004 The Thomson Corporation.  on STN
AN   2004:288652  BIOSIS
DN   PREV200400287409
TI   Expression of Myopodin Induces Suppression of Tumor Growth and Metastasis.
AU   Jing, Ling [Reprint Author]; Yu, Yan P; Luo, Jian-Hua
CS   Pathology, University of Pittsburgh, 3550 Terrace Street, Pittsburgh, PA,
     15261, USA
     luoj@msx.upmc.edu
SO   FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 619.9.
     http://www.fasebj.org/. e-file.
     Meeting Info.: FASEB Meeting on Experimental Biology: Translating the
     Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
     ISSN: 0892-6638 (ISSN print).
DT   Conference; (Meeting)
     Conference; Abstract; (Meeting Abstract)
LA   English
ED   Entered STN: 16 Jun 2004
     Last Updated on STN: 16 Jun 2004
AB   Myopodin was previously reported as a gene that was frequently deleted in
     prostate cancer.  This gene shares significant homology with a cell shape
     regulating gene, synaptopodin.  Myopodin was shown to bind actin, to
     induce actin bundling activity and to help forming stress fibers when
     cells were stimulated.  To clarify the functional role of myopodin in
     prostate cancer, several assays were performed to evaluate the tumor
     suppression activity of myopodin.  Our results indicate that myopodin
     inhibits tumor growth and invasion both in vitro and in
     vivo.  The activity of tumor suppression of myopodin is located at the
     C-terminus region.  To further evaluate the role of myopodin in developing
     invasiveness of prostate cancer, an expression analysis of myopodin
     protein was performed in prostate tissues.  The results indicate that
     down-regulation of myopodin expression occurs mostly in invasive stages of
     prostate cancer, implying a potential invasion suppression role for
     myopodin in prostate cancer.  In addition, hemizygous deletion and down
     regulation of myopodin expression occur in three aggressive prostate
     cancer cell lines.

L11  ANSWER 2 OF 3  CAPLUS  COPYRIGHT 2004 ACS on STN
AN   2003:478860  CAPLUS
DN   139:48251
TI   cDNA and protein sequences of a human cell adhesion inhibitory protein and
```

their use in diagnosis, therapy and drug screening
IN Ishikawa, Yoshinori; Goto, Masahiro; Sakamoto, Akihiro; Hirohashi, Setsuo
PA National Cancer Center, Japan
SO Jpn. Kokai Tokkyo Koho, 18 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2003174885	A2	20030624	JP 2001-378395	20011212
PRAI JP 2001-378395		20011212		

AB This invention provides cDNA and protein sequences of a human cell adhesion inhibitory protein. The protein was able to bind to actin and **inhibited** the cell adhesion of **cancer** cells. The protein shares sequence homol. with mouse gene RIC encoding protein and consists of signal peptide (residue 1-21), Thr-Ser-Pro rich extra membrane domain (residue 22-145), transmembrane domain (residue 146-162) and inner membrane domain (residue 163-178). The expression of the cell adhesion inhibitory protein was down regulated by cadherin. The protein can be used for diagnosis, **treatment** and screening drugs for **cancers**.

L11 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1996:312464 CAPLUS
DN 124:336063
TI Phosphorylation of human fascin inhibits its **actin binding** and bundling **activities**
AU Yamakita, Yoshihiko; Ono, Shoichiro; Matsumura, Fumio; Yamashiro, Shigeko
CS Dep. Mol. Biol. Biochem., Rutgers Univ., Piscataway, NJ, 08855-1059, USA
SO Journal of Biological Chemistry (1996), 271(21), 12632-12638
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB Human fascin (I) is an actin-bundling protein that is thought to be involved in the assembly of actin filament bundles present in microspikes as well as in membrane ruffles and stress fibers. Here, the authors found that human I is phosphorylated in vivo upon **treatment** with TPA, a **tumor** promoter. The in vivo phosphorylation was gradually increased from 0.13 to 0.30 mol/mol during 2 h of TPA treatment, concomitant with the disappearance of human I from stress fibers, membrane ruffles, and microspikes. Human I could also be phosphorylated in vitro by protein kinase C at the same sites as observed in vivo as judged by phosphopeptide mapping. The extent of phosphorylation depended on the pH: the stoichiometries were 0.05, 0.38, and 0.6 mol phosphate/mol protein at pH 7.0, 6.0, and 5.0, resp. Phosphorylation greatly reduced actin binding of human I, whereas lowering the pH to 6.0 alone did not affect I-actin binding. With the incorporation of 0.25 mol phosphate/mol I, the actin binding affinity was reduced from 6.7 + 106 to 1.5 + 106 M-1. The actin bundling activity was also decreased. These results suggest that phosphorylation of I plays a role in actin reorganization after treatment with TPA.

=> d bib ab 1-4 l12

L12 ANSWER 1 OF 4 MEDLINE on STN DUPLICATE 1
AN 2002283158 MEDLINE
DN PubMed ID: 12022372
TI Immunosuppressive and anticancer effect of a mammalian ribonuclease that targets high-affinity interleukin-2-receptors.
AU Yamamura Tadashi; Ueda Masakazu; Psarras Kyriakos; Suwa Tatsushi; Watanaabe Yasuo; Kameyama Noriaki; Tanabe Minoru; Imamura Hiroji; Kitajima

Masaki
CS Department of Surgery, Keio University School of Medicine, Tokyo, Japan.
SO European journal of surgery = Acta chirurgica, (2002) 168 (1) 49-54.
Journal code: 9105264. ISSN: 1102-4151.
CY Norway
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200211
ED Entered STN: 20020528
Last Updated on STN: 20021211
Entered Medline: 20021107
AB OBJECTIVE: To target high-affinity interleukin (IL)-2 receptors involved in lymphocyte proliferation processes such as allograft rejection, autoimmune disorders, and certain haematological malignancies, using a minimally immunogenic mammalian-derived enzyme, bovine RNaseA, which becomes cytotoxic on entering cytoplasm. DESIGN: Laboratory study. SETTING: Teaching hospital, Japan. MATERIAL: Human lymphocytes isolated from healthy histoincompatible donors in mixed lymphocyte cultures or stimulated with phytohemagglutinin (PHA) to promote IL-2Ralpha expression. MJ, an HTLV-1-infected malignant T-cell line that overexpresses IL-2Ralpha, and the IL-2Ralpha-negative cell lines MOLT-4F and MT-1, were used as controls. INTERVENTIONS: Bovine RNaseA was chemically conjugated to 7G7B6, a monoclonal antibody to the alpha-chain of human IL-2 receptors, and several concentrations of the conjugates were added to the lymphocyte cultures. MAIN OUTCOME MEASURES: Inhibition of cell proliferation as a percentage of 3H-thymidine incorporation in 24 hours. RESULTS: 7G7B6-RNaseA dose-dependently **inhibited** cell **proliferation** in PHA-stimulated human lymphocytes at a 50% inhibitory concentration (IC50) of 2 x 10(-7) **M**. whereas **RNase** alone and RNase plus antibody had no inhibitory effect. 7G7B6-RNaseA also dose-dependently inhibited the human mixed lymphocyte reaction at an IC50 of 2 x 10(-6) **M**, whereas **RNase** alone did not. The conjugate also inhibited cell proliferation in MJ cells, a cell line that is infected with HTLV-I and overexpresses the high-affinity IL-2 receptor, at an IC50 of 5 x 10(-7) M. However the conjugate had no inhibitory effect on the IL-2 receptor non-expressing human T-cell lymphoblastic leukaemia cell lines MOLT-4F or MT-1. CONCLUSION: 7G7B6-RNaseA can inhibit cell proliferation in antigen- or mitogen-stimulated lymphocytes that overexpress high-affinity IL-2 receptors, and it may be safer than conventional chemotherapy or immunotoxins in the treatment of transplant rejection, certain lymphocytic malignancies, and other IL-2R-associated diseases, because it contains a mammalian cytotoxic enzyme.

L12 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:167748 CAPLUS
DN 134:188197
TI Methods of and compositions for inhibiting the proliferation of mammalian cells
IN Roiz, Levava; Schwartz, Betty; Smirnoff, Patricia; Shoseyov, Oded
PA Yissum Research Development Company of the Hebrew University of Jerusalem, Israel
SO PCT Int. Appl., 91 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001015531	A1	20010308	WO 2000-IL514	20000829

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,

LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2382303	AA	20010308	CA 2000-2382303	20000829
EP 1207755	A1	20020529	EP 2000-954879	20000829
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003508411	T2	20030304	JP 2001-519760	20000829
AU 769011	B2	20040115	AU 2000-67228	20000829
NZ 517579	A	20040227	NZ 2000-517579	20000829
ZA 2002001647	A	20030310	ZA 2002-1647	20020227
PRAI US 1999-385411	A	19990830		
WO 2000-IL514	W	20000829		

AB A method of preventing, inhibiting and/or reversing proliferation, colonization, differentiation and/or development of abnormally proliferating cells in a subject is disclosed. The method is effected by administering to the subject a therapeutically effective amount of a RNase of the T2 family.

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 4 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2001:855636 SCISEARCH
GA The Genuine Article (R) Number: 483NK
TI Inhibition of human brain tumor cell growth by the anti-inflammatory drug, flurbiprofen
AU King J G; Khalili K (Reprint)
CS Temple Univ, Coll Sci & Technol, Ctr Neurovirol & Canc Biol, Lab Canc Biol & Intervent, 1900 N 12th St, 015-96, Room 203, Philadelphia, PA 19122 USA (Reprint); Temple Univ, Coll Sci & Technol, Ctr Neurovirol & Canc Biol, Lab Canc Biol & Intervent, Philadelphia, PA 19122 USA
CYA USA
SO ONCOGENE, (18 OCT 2001) Vol. 20, No. 47, pp. 6864-6870.
Publisher: NATURE PUBLISHING GROUP, HOUNDMILLS, BASINGSTOKE RG21 6XS, HAMPSHIRE, ENGLAND.
ISSN: 0950-9232.
DT Article; Journal
LA English
REC Reference Count: 24
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Despite many efforts to alter the relentlessly aggressive progression of tumors of neural origin, individuals bearing these tumors exhibit poor prognosis for longterm survival. In an attempt to find an effective treatment, we examined the efficacy of the non-steroidal anti-inflammatory drug, flurbiprofen, to suppress the growth of tumor cell lines derived from medulloblastoma and glioblastoma multiforme. Results from cell proliferation assays have revealed that flurbiprofen effectively **inhibits** the growth of various **tumor** cells in a dose-dependent manner and causes a noticeable change in the progression of cells through cell cycle stages. **Treatment** of **tumor** cells with flurbiprofen **reduced** the number of cells in G1 and G2, and significantly increased their numbers in S phase, suggesting that, flurbiprofen accelerates G1/S entry, and/or delays cell exit from S to G2/**M** stages. Results from **RNase** protection assay and Western blot analysis showed that while treatment of cells with flurbiprofen causes a minor change in the RNA level of different cyclins, there is a significant decrease in the level of cyclin B protein upon flurbiprofen **treatment**. Examination of **tumor** suppressors by RNase protection technique showed a subtle increase in the levels of several **tumor** suppressors upon flurbiprofen

treatment. Interestingly, at the protein level, p53 tumor suppressor was substantially increased upon flurbiprofen treatment, yet the level of p21, a downstream target for p53 remained unchanged. Curiously, treatment of the cells with flurbiprofen enhanced the level of COX-2 expression. Results from co-immunoprecipitation showed association of COX-2 with p53 in tumor cells. These observations suggest that the interaction of COX-2 with p53 may cause p21-independent suppression of **tumor** cell growth upon flurbiprofen **treatment**.

L12 ANSWER 4 OF 4 MEDLINE on STN DUPLICATE 2
AN 77023708 MEDLINE
DN PubMed ID: 975050
TI Comparison of antitumor activities of pancreatic ribonuclease and its cross-linked dimer.
AU Tarnowski G S; Kassel R L; Mountain I M; Blackburn P; Wilson G; Wang D
SO Cancer research, (1976 Nov) 36 (11 Pt 1) 4074-8.
Journal code: 2984705R. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197612
ED Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19761230
AB The cross-linked dimer of bovine pancreatic **RNase** (**M**.W. 28,000) is significantly more effective than the monomer in **inhibiting tumor** development in mice when administered i.p. 1 day after inoculation with sarcoma 180J ascites cells. Animals bearing solid tumors were not affected. In AKR/J mice with advanced leukemia, a single i.p. injection of 100 mug of the dimer led to about 50% reduction in the enlarged lymph nodes and the spleen at 24 hr. The half-life of the dimer in the bloodstream has been determined to be 10 min in rats and 6 min in mice, compared to values of 5 and 3.5 min, respectively, for the monomer. Analyses of the tissues of untreated leukemic mice for RNase and RNase inhibitors show that the tumor tissues are not deficient in RNase activity. Considerations of possible mechanisms of action of the dimer indicate that other basic proteins in this size range may merit examination as cytostatic agents toward transformed cells.

=> d bib ab 1-2 l13

L13 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2004:791880 CAPLUS
DN 141:364076
TI Comparative study of proteome between primary cancer and hepatic metastatic tumor in colorectal cancer
AU Yu, Bo; Li, Shi-Yong; An, Ping; Zhang, Ying-Nan; Liang, Zhen-Jia; Yuan, Shu-Jun; Cai, Hui-Yun
CS Department of General Surgery, General Hospital of Beijing Military Command, Beijing, 100700, Peop. Rep. China
SO World Journal of Gastroenterology (2004), 10(18), 2652-2656
CODEN: WJGAF2; ISSN: 1007-9327
PB World Journal of Gastroenterology
DT Journal
LA English
AB AIM: To identify the differential proteins associated with colorectal cancer genesis and hepatic metastasis. METHODS: Hydrophobic protein samples were extracted from normal colorectal mucosa, primary cancer lesion and hepatic metastatic foci of colorectal cancer. With two-dimensional electrophoresis and image anal., differentially expressed protein spots were detected, and the proteins were identified by matrix assisted laser

desorption/ionization-time of flight-mass spectrometry and peptide mass fingerprint anal. RESULTS: Significant alterations of the proteins in number and expression levels were discovered in primary cancer and hepatic metastatic foci, the expression of a number of proteins was lost in 25-40 ku, but protein spots were increased in 14-21 ku, compared with normal mucosa. Nine differentially expressed protein spots were identified. Three proteins expressed in normal mucosa, but lost in primary cancer and hepatic metastasis, were recognized as calmodulin, RNase 6 precursor and mannosidase-α. Proapolipoprotein was expressed progressively from normal mucosa to primary cancer and hepatic metastasis. The differentially expressed protein of beta-globin was found in normal mucosa and hepatic metastatic tumor, but lost in primary cancer lesion. Cdc42, a GTP-binding protein, was identified in hepatic metastasis. The protein spots of C4 from primary cancer, M7 and M9 from hepatic metastasis had less homol. with the proteins in database. CONCLUSION: Variations of hydrophobic protein expression in colorectal cancer initiation and hepatic metastasis are significant and can be observed with two-dimensional electrophoresis. The expression of calmodulin, RNase 6 precursor and mannosidase-α is lost but the expression of proapolipoprotein is **enhanced** which is associated with colorectal **cancer** genesis and hepatic metastasis. Cdc 42 and beta-globin are expressed abnormally in hepatic metastasis. Protein C4, M7 and M9 may be associated with colorectal cancer genesis and hepatic metastasis.

RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 2 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2001:855636 SCISEARCH
GA The Genuine Article (R) Number: 483NK
TI Inhibition of human brain tumor cell growth by the anti-inflammatory drug, flurbiprofen
AU King J G; Khalili K (Reprint)
CS Temple Univ, Coll Sci & Technol, Ctr Neurovirol & Canc Biol, Lab Canc Biol & Intervent, 1900 N 12th St, 015-96, Room 203, Philadelphia, PA 19122 USA (Reprint); Temple Univ, Coll Sci & Technol, Ctr Neurovirol & Canc Biol, Lab Canc Biol & Intervent, Philadelphia, PA 19122 USA
CYA USA
SO ONCOGENE, (18 OCT 2001) Vol. 20, No. 47, pp. 6864-6870.
Publisher: NATURE PUBLISHING GROUP, HOUNDMILLS, BASINGSTOKE RG21 6XS, HAMPSHIRE, ENGLAND.
ISSN: 0950-9232.
DT Article; Journal
LA English
REC Reference Count: 24
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Despite many efforts to alter the relentlessly aggressive progression of tumors of neural origin, individuals bearing these tumors exhibit poor prognosis for longterm survival. In an attempt to find an effective treatment, we examined the efficacy of the non-steroidal anti-inflammatory drug, flurbiprofen, to suppress the growth of tumor cell lines derived from medulloblastoma and glioblastoma multiforme. Results from cell proliferation assays have revealed that flurbiprofen effectively inhibits the growth of various tumor cells in a dose-dependent manner and causes a noticeable change in the progression of cells through cell cycle stages. Treatment of tumor cells with flurbiprofen reduced the number of cells in G1 and G2, and significantly increased their numbers in S phase, suggesting that, flurbiprofen accelerates G1/S entry, and/or delays cell exit from S to G2/**M** stages. Results from **RNase** protection assay and Western blot analysis showed that while treatment of cells with flurbiprofen causes a minor change in the RNA level of different cyclins, there is a significant decrease in the level of cyclin B protein upon flurbiprofen treatment. Examination of tumor suppressors by RNase protection technique showed a subtle **increase** in the

levels of several **tumor** suppressors upon flurbiprofen treatment. Interestingly, at the protein level, p53 **tumor** suppressor was substantially **increased** upon flurbiprofen treatment, yet the level of p21, a downstream target for p53 remained unchanged. Curiously, treatment of the cells with flurbiprofen enhanced the level of COX-2 expression. Results from co-immunoprecipitation showed association of COX-2 with p53 in tumor cells. These observations suggest that the interaction of COX-2 with p53 may cause p21-independent suppression of tumor cell growth upon flurbiprofen treatment.

=> d bib ab l14

L14 ANSWER 1 OF 1 MEDLINE on STN DUPLICATE 1
AN 96218192 MEDLINE
DN PubMed ID: 8647875
TI Phosphorylation of human fascin inhibits its **actin binding** and bundling **activities**.
AU Yamakita Y; Ono S; Matsumura F; Yamashiro S
CS Department of Molecular Biology and Biochemistry, Rutgers University, Piscataway, New Jersey 08855-1059, USA.
NC R37 CA42742 (NCI)
SO Journal of biological chemistry, (1996 May 24) 271 (21) 12632-8.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199607
ED Entered STN: 19960805
Last Updated on STN: 19990129
Entered Medline: 19960722
AB Human fascin is an actin-bundling protein that is thought to be involved in the assembly of actin filament bundles present in microspikes as well as in membrane ruffles and stress fibers. We have found that human fascin is phosphorylated in vivo upon treatment with 12-O-tetradecanoylphorbol-13-acetate, a **tumor promoter**. The in vivo phosphorylation is gradually increased from 0.13 to 0.30 mol/mol during 2 h of treatment, concomitant with disappearance of human fascin from stress fibers, membrane ruffles, and microspikes. Human fascin can also be phosphorylated in vitro as judged by phosphopeptide mapping. The extent of phosphorylation depends on pH: the stoichiometries are 0.05, 0.38, and 0.6 alone does not affect fascin-actin binding. With the incorporation of 0.25 mol of phosphate/mol of protein, the actin binding affinity is reduced from 6.7 x 10(6) to 1.5 x 10(6) m(-1). The actin bundling activity is also decreased. These results suggest that phosphorylation of fascin plays a role in actin reorganization after treatment with 12-O-tetradecanoylphorbol-13-acetate.

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     (FILE 'HOME' ENTERED AT 15:07:47 ON 22 DEC 2004)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 15:08:15 ON 22 DEC
     2004
L1          1454 S RNASE(3A)(T2 OR RH OR M OR TRV OR IRP OR LE OR MC1 OR TP OR O
L2           813 S ACTIN(W)BIND?(3A)ACTIVITY
L3             0 S L1 AND L2
L4        635993 S (INHIBIT? OR REDUC? OR PREVENT? OR TREAT? OR REVERS?)(5A)(CAN
L5             7 S L1(S)L4
L6             0 S L2(S)L4
L7             3 S L2 AND L4
L8        337629 S (PROMOT? OR INCREAS? OR FACILITAT? OR ENHANC?)(6A)(CANCER OR
L9             2 S L1 AND L8
L10            4 S L2 AND L8
L11            3 DUP REM L7 (0 DUPLICATES REMOVED)
L12            4 DUP REM L5 (3 DUPLICATES REMOVED)
L13            2 DUP REM L9 (0 DUPLICATES REMOVED)
L14            1 DUP REM L10 (3 DUPLICATES REMOVED)
L15      3189813 S PROBLEM OR DEFECT OR DIFFICULT? OR DRAWBACK
L16         9386 S L4(S)L15
L17        67390 S (PROTEIN OR POLYPEPTIDE OR RNASE)(3A)(TREAT? OR THERAPY)
L18           46 S L16 AND L17
L19            6 S REVIEW AND L18
L20           36 DUP REM L18 (10 DUPLICATES REMOVED)
L21            6 DUP REM L19 (0 DUPLICATES REMOVED)

=> d au ti so pi ab 1-6 l21

L21  ANSWER 1 OF 6  SCISEARCH  COPYRIGHT (c) 2004 The Thomson Corporation.  on
     STN
AU   Bokemeyer C (Reprint); Aapro M S; Courdi A; Foubert J; Link H; Osterborg
     A; Repetto L; Soubeyran P
TI   EORTC guidelines for the use of erythropoietic proteins in anaemic
     patients with cancer
SO   EUROPEAN JOURNAL OF CANCER, (OCT 2004) Vol. 40, No. 15, pp. 2201-2216.
     Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE,
     KIDLINGTON, OXFORD OX5 1GB, ENGLAND.
     ISSN: 0959-8049.
AB       Anaemia is frequently diagnosed in patients with cancer, yet it is
     difficult to identify a single cause due to its multifactorial
     aetiology. We conducted a systematic literature review
     (1996-2003) to produce evidence-based guidelines on the use of
     erythropoietic proteins in anaemic patients with cancer (see Table 4).
     Level I evidence exists for a positive impact of erythropoietic proteins
     on haemoglobin (Hb) levels when administered to patients with
     chemotherapy-induced anaemia or anaemia of chronic disease, when used to
     prevent cancer anaemia, in patients undergoing
     cancer surgery and following allogeneic bone marrow
     transplantation. The Hb level at which erythropoietic protein
     therapy should be initiated is difficult to determine as
     it varied between studies; a large number of Level I studies in patients
     with chemotherapy-induced anaemia or anaemia of chronic disease enrolled
     patients with a Hb concentration less than or equal to105 g/L, but none
     compared the effect of different baseline Hb levels on the response to
     treatment. Similarly, several studies defined the target Hb concentration
     as 120-130 g/L following treatment with erythropoietic
     proteins, but none specifically addressed the correlation between
     target Hb level and clinical benefit in a randomised fashion. Level I
     evidence shows that red blood cell (RBC) transfusion requirements are
     significantly reduced with erythropoietic protein
     therapy in patients with chemotherapy-induced anaemia or when used
     to prevent cancer anaemia (approximately 20%
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reduction compared with controls). We found indirect Level 1 and III evidence that patients with chemotherapy-induced anaemia or anaemia of chronic disease initially classified as non-responders to standard doses proceed to respond to treatment following a dose increase (absolute increases in response rate ranged from 8% to 18%). However, none of these studies examined the effect on response rates of a longer treatment period at the lower dose, or performed a randomised comparison of a dose increase versus an unchanged dose. There is Level I evidence to show that quality-of-life (QOL) is significantly improved in patients with chemotherapy-induced anaemia and in those with anaemia of chronic disease, particularly in patients achieving a Hb response to erythropoietic **protein therapy**. There are insufficient data to determine the effect on survival following **treatment** with erythropoietic **proteins** in conjunction with chemotherapy or radiotherapy. There is Level I evidence that dosing of erythropoietic proteins less frequently than three times per week (TIW) is efficacious when used to **treat** chemotherapy-induced anaemia or **prevent cancer** anaemia. There is Level III evidence that initial doses of erythropoietic proteins considered to be higher than current standard practice produce higher haematological responses in patients with chemotherapy-induced anaemia or anaemia of chronic disease. Level I evidence demonstrates that several baseline patient parameters (e.g., low endogenous erythropoietin [EPO] concentration, age <60 years, Hb concentration greater than or equal to 90 g/L) impact upon the response to erythropoietic **proteins** when used to **treat** chemotherapy-induced anaemia or **prevent cancer** anaemia. Evidence indicates that endogenous EPO concentration impacts on response in patients with lymphoproliferative malignancies, but is not a valid parameter in patients with solid tumours.

There is Level I evidence that fixed doses of erythropoietic proteins can be used at the start of therapy to treat patients with chemotherapy-induced anaemia, but maintenance doses should be titrated individually. There is no evidence that pure red cell aplasia (PRCA) occurs following **treatment** with erythropoietic **proteins** in patients with chemotherapy-induced anaemia or when used prophylactically in patients with cancer. There is Level I evidence that the risk of thromboembolic events and hypertension are slightly elevated in patients with chemotherapy-induced anaemia receiving erythropoietic proteins. Level I evidence supports the effectiveness of erythropoietic proteins to prevent anaemia in non-anaemic cancer patients receiving chemotherapy or radiotherapy or in those undergoing cancer surgery. However, these are non-licensed indications and we do not currently recommend the prophylactic use of erythropoietic proteins to prevent anaemia in patients who have normal Hb values at the start of treatment.

Additional trials are warranted, especially on the issues of iron replacement and cost-effectiveness of erythropoietic **protein therapy**, as well as on tumour response/progression and survival.

L21 ANSWER 2 OF 6 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

AU Oro C (Reprint); Jans D A

TI The tumour specific pro-apoptotic factor apoptin (Vp3) from chicken anaemia virus

SO CURRENT DRUG TARGETS, (FEB 2004) Vol. 5, No. 2, pp. 179-190.
Publisher: BENTHAM SCIENCE PUBL LTD, PO BOX 1673, 1200 BR HILVERSUM, NETHERLANDS.
ISSN: 1389-4501.

AB Cancer is a growing **problem** for human health world-wide. Dramatic breakthroughs have increased our understanding of the molecular mechanisms involved in the process of tumorigenesis, allowing us to develop more refined anti-**cancer treatments**, expanding the repertoire of available anti-cancer drugs, and increasing the efficiency of their delivery to malignant cells. Nevertheless, even with

improved understanding of the complex origins of cancer cells, there is a dearth of efficient and above all specific anti-**cancer treatments**. Apoptin (viral **protein** 3 - VP3), a gene product derived from the Chicken Anaemia Virus (CAV) represents a novel anti-cancer tool. It appears to have innate tumour-specific p53 -independent, Bcl-2-enhanced proapoptotic activity, and hence may be of great utility in the endeavour to achieve specific and efficient elimination of cancer cells, particularly in cases of drug resistance through Bcl-2 overexpression/loss of p53 function etc. This **review** will examine the unique aspects of apoptin's properties, and in particular, its ability to localise specifically in the nucleus of transformed but not normal cells. The latter ability, importantly, appears to be integrally related to its tumour-specific pro-apoptotic action.

L21 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AU Body, Jean-Jacques; Mancini, Isabelle
TI **Treatment** of **tumor**-induced hypercalcemia: a solved **problem**?
SO Expert Review of Anticancer Therapy (2003), 3(2), 241-246
CODEN: ERATBJ; ISSN: 1473-7140
AB A **review**. Less than 25 yr ago, tumor-induced hypercalcemia was often a lethal complication of cancer. Nowadays, it can be successfully and easily treated in at least 90% of the cases by rehydration and potent antiosteoclastic bisphosphonates. The standard therapy consists of the administration of 90 mg of pamidronate (Aredia Dry Powder) or more recently, 4 mg of zoledronic acid (Zometa), which is even more efficient, at least in patients without bone metastases. Recurrent hypercalcemia is nevertheless difficult to control and antibodies against parathyroid-hormone-related protein may prove to be a useful treatment.

L21 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AU Li, Han; Pamukcu, Rifat; Thompson, W. Joseph
TI β-Catenin signaling: therapeutic strategies in oncology
SO Cancer Biology & Therapy (2002), 1(6), 621-625
CODEN: CBTAAO; ISSN: 1538-4047
AB A **review**. Activated Wnt signaling pathways have been found in various human cancers, including those of the colon, liver, endometrium, ovary, prostate, and stomach. As a result, β-catenin is accumulated and becomes transcriptionally active for proliferative genes and oncogenes. Wnt pathway mutations result in biochem. mechanisms yielding inefficient phosphorylation of β-catenin by glycogen synthase kinase 3β (GSK3β) due to APC, β-catenin and/or axin mutations. Therefore, the needs and the opportunity to develop new cancer therapies exist through reversing oncogenic APC/β-catenin/Lef/Tcf signals. Exisulind and analogs are inhibitors of cyclic GMP phosphodiesterases (PDE) that have been shown to activate and induce protein kinase G. The data show PKG regulation of β-catenin in Wnt signaling, accounting, at least in part, for apoptosis induction in treated colon cancer cells carrying either APC or β-catenin mutations. Exisulind and analogs reduce β-catenin via a novel, GSK3β independent processing mechanism. Activated PKG directly phosphorylate β-catenin at its C-terminal domain and causes proteasome dependent degradation of the protein. Since this pathway is independent of APC and GSK3β, exisulind and analogs provide a superior approach to circumvent the mol. **defects** of Wnt signaling pathway and to **treat cancers** with such **defects**.

L21 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AU Sachdeva, Mandip Singh
TI Drug targeting systems for cancer chemotherapy
SO Expert Opinion on Investigational Drugs (1998), 7(11), 1849-1864
CODEN: EOIDER; ISSN: 1354-3784
AB A **review** with 224 refs. Tumor specific drug targeting has been a very actively investigated area for over 2 decades. Various approaches

have involved the use of drug delivery systems that can localize the anticancer agent at the tumor site without damaging the normal cells. For this purpose, various delivery systems than have been utilized are liposomes, microspheres and recently, nanoparticles. Two liposome formulations containing anticancer drugs for example, adriamycin and daunomycin are already on the market in the USA and Europe. Microspheres are also being investigated for delivering various anticancer drugs and **protein**/peptides for anticancer **treatment**, and several formulations are in Phase I/II clin. trials. Antitumor drugs have also been linked to tumor specific monoclonal antibodies via various chemical linkages. Doxorubicin was linked to a chimeric monoclonal antibody that was targeted to the Lewis Y antigen. Though this conjugate initially showed potential it was recently dropped from Phase II clin. trials. Another approach with monoclonal antibodies has been the use of immunotoxins. Immunotoxins initially showed promise as potential anticancer agents at picomolar concns. but several clin. and preclin. studies have not shown much promise in this regard. Drug containing liposomes and microspheres have been further linked to tumor specific monoclonal antibodies to enhance their tumor specificity. Most of the studies with immunoliposomes or targeted microspheres have not gone beyond the preclin. studies. New therapeutic approaches are presently emerging based on natural products like cytokines, peptide growth factor antagonists, antisense oligonucleotides and specific genes. These approaches need the help of delivery systems to deliver these complex mols. to tumor cells. One of the current pursued approaches in the use of cationic liposomes. Several clin. studies are undergoing with various cationic liposomes and the next rew years will demonstrate the usefulness of this approach. In recent years, the **problems** in **cancer treatment** have been complicated with the emergence of resistance strains leading to resistant and cross-resistant tumor cells. Several agents have been used to overcome or reverse drug-resistance in solid tumors and it remains a highly pursued area in cancer treatment.

L21 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AU Nishimoto, Ikuo
TI G protein regulation in the strategy to create disease therapy
SO Horumon to Rinsho (1997), 45(12), 1131-1139
CODEN: HORIAE; ISSN: 0045-7167
AB A **review** with 35 refs., on pathophysiol. of G protein diseases including diseases caused by **defects** in G proteins and G protein-coupled receptors, G protein-targeted therapeutic strategy, and therapeutic strategy using G protein-mediated signaling pathway, e.g., **treatment** of endocrine **tumors** with somatostatin by proliferation suppression and apoptosis induction.

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     (FILE 'HOME' ENTERED AT 16:14:20 ON 22 DEC 2004)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 16:14:38 ON 22 DEC
     2004
L1       124517 S RNASE OR RIBONUCLEASE
L2          563 S ACTIN(W)BIND?(W)ACTIVITY
L3          813 S ACTIN(W)BIND?(3A)ACTIVITY
L4       337629 S (PROMOT? OR INCREAS? OR FACILITAT? OR ENHANC?)(6A)(CANCER OR
L5            0 S L1 AND L2
L6            0 S L1 AND L3
L7           91 S L1(7A)L4
L8        16731 S ACTIN(W)BIND?
L9           46 S L1 AND L8
L10           2 S L9 AND L4
L11        1461 S RNASE(3A)(T2 OR RH OR M OR TRV OR IRP OR LE OR MC1 OR TP OR O
L12           0 S L11 AND L8
L13          24 DUP REM L9 (22 DUPLICATES REMOVED)
L14           2 DUP REM L10 (0 DUPLICATES REMOVED)

=> d au ti so pi ab 1-2 l14

L14  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2004 ACS on STN
IN   Katagiri, Toyomasa; Ohnishi, Yasuyuki; Nakamura, Yusuke
TI   Genetic cancer profiles for drug screening and personalized cancer
     treatment
SO   PCT Int. Appl., 76 pp.
     CODEN: PIXXD2
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
PI   WO 2003057916        A2    20030717    WO 2003-IB360           20030109
     WO 2003057916        A3    20040422
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     US 2003165954        A1    20030904    US 2003-339533          20030109
     EP 1466016           A2    20041013    EP 2003-700442          20030109
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
AB   The invention relates to genetic profiles and markers of cancers and
     provides systems and methods for screening drugs that are effective for
     specific patients and types of cancers.  In particular, the invention
     provides personalized treatment customized to an individual's cancer.

L14  ANSWER 2 OF 2  CAPLUS  COPYRIGHT 2004 ACS on STN
AU   Zhang, Jianzhi; Rosenberg, Helene F.
TI   Diversifying selection of the tumor-growth promoter
     angiogenin in primate evolution
SO   Molecular Biology and Evolution (2002), 19(4), 438-445
     CODEN: MBEVEO; ISSN: 0737-4038
AB   Diversifying selection drives the rapid differentiation of gene sequences
     and is one of the main forces behind adaptive evolution.  Most genes known
     to be shaped by diversifying selection are those involved in host-pathogen
     or male-female interactions characterized as mol. "arms races.".  Here we
     report the unexpected detection of diversifying selection in the evolution
     of a tumor-growth promoter, angiogenin (ANG).  A
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comparison among 11 primate species demonstrates that ANG has a significantly higher rate of nucleotide substitution at nonsynonymous sites than at synonymous sites, a hallmark of pos. selection acting at the mol. level. Furthermore, we observed significant charge diversity at the mol. surface, suggesting the presence of selective pressures in the microenvironment of ANG, including its binding mols. A population survey of ANG in chimpanzees, however, reveals no polymorphism, which may have resulted from a recent selective sweep of a charge-altering substitution in chimpanzee evolution. Functional assays of recombinant ANGs from the human and owl monkey indicate that primate ANGs retain angiogenic activity despite rapid evolution. Our study, together with findings of similar selection in the primate breast cancer suppressor gene, BRCA1, reveals an intriguing phenomenon of unusual selective pressures on, and adaptive evolution of, cancer-related genes in primate evolution.

=> d au ti so 1-24 l13

L13 ANSWER 1 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
IN Erlander, Mark G.; Ma, Xiao-Jun; Wang, Wei; Wittliff, James L.
TI DNA microarray analysis of gene expression in the diagnosis of estrogen receptor positive- and negative-breast cancer
SO PCT Int. Appl., 226 pp.
CODEN: PIXXD2

L13 ANSWER 2 OF 24 MEDLINE on STN DUPLICATE 1
AU Zhang Suisheng; Kohler Carsten; Hemmerich Peter; Grosse Frank
TI Nuclear DNA helicase II (RNA helicase A) binds to an F-actin containing shell that surrounds the nucleolus.
SO Experimental cell research, (2004 Feb 15) 293 (2) 248-58.
Journal code: 0373226. ISSN: 0014-4827.

L13 ANSWER 3 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
IN Katagiri, Toyomasa; Ohnishi, Yasuyuki; Nakamura, Yusuke
TI Genetic cancer profiles for drug screening and personalized cancer treatment
SO PCT Int. Appl., 76 pp.
CODEN: PIXXD2

L13 ANSWER 4 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
IN Hung, David T.
TI Identifying material from a breast duct
SO U.S., 19 pp., Cont.-in-part of U.S. Ser. No. 502,404.
CODEN: USXXAM

L13 ANSWER 5 OF 24 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Roiz L (Reprint); Smirnoff P; Shoseyov O; Schwartz B
TI Actibind: An **actin-binding** fungal T-2-**RNase** with anticancer effect.
SO CLINICAL CANCER RESEARCH, (1 DEC 2003) Vol. 9, No. 16, Part 2, Supp. [S], pp. 6149S-6149S.
Publisher: AMER ASSOC CANCER RESEARCH, 615 CHESTNUT ST, 17TH FLOOR, PHILADELPHIA, PA 19106-4404 USA.
ISSN: 1078-0432.

L13 ANSWER 6 OF 24 MEDLINE on STN DUPLICATE 2
AU Gho Yong Song; Yoon Wan-Hee; Chae Chi-Bom
TI Antiplasmin activity of a peptide that binds to the receptor-binding site of angiogenin.
SO Journal of biological chemistry, (2002 Mar 22) 277 (12) 9690-4.
Journal code: 2985121R. ISSN: 0021-9258.

L13 ANSWER 7 OF 24 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on

STN
AU Vugmeyster L; Trott O; McKnight C J; Raleigh D P (Reprint); Palmer A G
TI Temperature-dependent dynamics of the villin headpiece helical subdomain, an unusually small thermostable protein
SO JOURNAL OF MOLECULAR BIOLOGY, (19 JUL 2002) Vol. 320, No. 4, pp. 841-854.
Publisher: ACADEMIC PRESS LTD ELSEVIER SCIENCE LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND.
ISSN: 0022-2836.

L13 ANSWER 8 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
AU Zhang, Jianzhi; Rosenberg, Helene F.
TI Diversifying selection of the tumor-growth promoter angiogenin in primate evolution
SO Molecular Biology and Evolution (2002), 19(4), 438-445
CODEN: MBEVEO; ISSN: 0737-4038

L13 ANSWER 9 OF 24 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Silva N F; Goring D R (Reprint)
TI Mechanisms of self-incompatibility in flowering plants
SO CELLULAR AND MOLECULAR LIFE SCIENCES, (DEC 2001) Vol. 58, No. 14, pp. 1988-2007.
Publisher: BIRKHAUSER VERLAG AG, VIADUKSTRASSE 40-44, PO BOX 133, CH-4010 BASEL, SWITZERLAND.
ISSN: 1420-682X.

L13 ANSWER 10 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
IN Hung, David T.
TI Using markers for the identification of breast cancer and precancer from breast duct samples
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2

L13 ANSWER 11 OF 24 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Schelp C (Reprint); GreiserWilke I; Moennig V
TI An **actin-binding** protein is involved in pestivirus entry into bovine cells
SO VIRUS RESEARCH, (JUN 2000) Vol. 68, No. 1, pp. 1-5.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0168-1702.

L13 ANSWER 12 OF 24 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Yokoro K; Yanagidani A; Obata T; Yamamoto S; Numoto M (Reprint)
TI Genomic cloning and characterization of the mouse POZ/zinc-finger protein ZF5
SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (29 MAY 1998) Vol. 246, No. 3, pp. 668-674.
Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0006-291X.

L13 ANSWER 13 OF 24 MEDLINE on STN DUPLICATE 3
AU Bies R D; Maeda M; Roberds S L; Holder E; Bohlmeyer T; Young J B; Campbell K P
TI A 5' dystrophin duplication mutation causes membrane deficiency of alpha-dystroglycan in a family with X-linked cardiomyopathy.
SO Journal of molecular and cellular cardiology, (1997 Dec) 29 (12) 3175-88.
Journal code: 0262322. ISSN: 0022-2828.

L13 ANSWER 14 OF 24 MEDLINE on STN
AU Choi S J; Ahn M; Lee J S; Jung W J

TI Selection of a high affinity angiogenin-binding peptide from a peptide library displayed on phage coat protein.
SO Molecules and cells, (1997 Oct 31) 7 (5) 575-81.
Journal code: 9610936. ISSN: 1016-8478.

L13 ANSWER 15 OF 24 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AU Lallena M J; Correas I (Reprint)
TI Transcription-dependent redistribution of nuclear protein 4.1 to SC35-enriched nuclear domains
SO JOURNAL OF CELL SCIENCE, (JAN 1997) Vol. 110, Part 2, pp. 239-247.
Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE, CAMBS, ENGLAND CB4 4DL.
ISSN: 0021-9533.

L13 ANSWER 16 OF 24 MEDLINE on STN DUPLICATE 4
AU Elliott C E; Becker B; Oehler S; Castanon M J; Hauptmann R; Wiche G
TI Plectin transcript diversity: identification and tissue distribution of variants with distinct first coding exons and rodless isoforms.
SO Genomics, (1997 May 15) 42 (1) 115-25.
Journal code: 8800135. ISSN: 0888-7543.

L13 ANSWER 17 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 5
AU Chang, Soo-Ik; Paik, Seung-Bum; So, Seung-Ho; Ahn, Byung-Cheol
TI Interaction between a blood vessel-inducing protein angiogenin and its binding protein actin
SO Journal of Biochemistry and Molecular Biology (1996), 29(4), 353-358
CODEN: JBMBE5; ISSN: 1225-8687

L13 ANSWER 18 OF 24 MEDLINE on STN DUPLICATE 6
AU Temm-Grove C J; Guo W; Helfman D M
TI Low molecular weight rat fibroblast tropomyosin 5 (TM-5): cDNA cloning, **actin-binding**, localization, and coiled-coil interactions.
SO Cell motility and the cytoskeleton, (1996) 33 (3) 223-40.
Journal code: 8605339. ISSN: 0886-1544.

L13 ANSWER 19 OF 24 MEDLINE on STN DUPLICATE 7
AU Kondo T; Shirasawa T; Itoyama Y; Mori H
TI Embryonic genes expressed in Alzheimer's disease brains.
SO Neuroscience letters, (1996 May 17) 209 (3) 157-60.
Journal code: 7600130. ISSN: 0304-3940.

L13 ANSWER 20 OF 24 MEDLINE on STN DUPLICATE 8
AU Nefsky B; Bretscher A
TI Preparation of immobilized monomeric actin and its use in the isolation of protease-free and **ribonuclease**-free pancreatic deoxyribonuclease I.
SO European journal of biochemistry / FEBS, (1989 Jan 15) 179 (1) 215-9.
Journal code: 0107600. ISSN: 0014-2956.

L13 ANSWER 21 OF 24 MEDLINE on STN
AU Kwiatkowski D J; Mehl R; Izumo S; Nadal-Ginard B; Yin H L
TI Muscle is the major source of plasma gelsolin.
SO Journal of biological chemistry, (1988 Jun 15) 263 (17) 8239-43.
Journal code: 2985121R. ISSN: 0021-9258.

L13 ANSWER 22 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
AU Scheer, Ulrich; Hinssen, Horst; Franke, Werner W.; Jockusch, Brigitte M.
TI Microinjection of **actin-binding** proteins and actin antibodies demonstrates involvement of nuclear actin in transcription of lampbrush chromosomes
SO Cell (Cambridge, MA, United States) (1984), 39(1), 111-22
CODEN: CELLB5; ISSN: 0092-8674

L13 ANSWER 23 OF 24 MEDLINE on STN DUPLICATE 9
AU Griffith L M; Pollard T D
TI Cross-linking of actin filament networks by self-association and **actin-binding** macromolecules.
SO Journal of biological chemistry, (1982 Aug 10) 257 (15) 9135-42.
Journal code: 2985121R. ISSN: 0021-9258.

L13 ANSWER 24 OF 24 CAPLUS COPYRIGHT 2004 ACS on STN
AU Baril, Earl F.; Herrmann, Heinz
TI Muscle development. II. Immunological and enzymic properties and accumulation of chromatographically homogeneous myosin of the leg musculature of the developing chick
SO Developmental Biology (Orlando, FL, United States) (1967), 15(4), 318-33
CODEN: DEBIAO; ISSN: 0012-1606